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Vol. XXV MARCH 1959 No. 9

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COMMON DISEASES OF THE LIVER

By

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It is fortunate that in most diseased states of the liver the organ becomes enlarged and enlarged downwards so that it becomes palpable. The doctor practising in the tropics should be particularly capable of recognising the least palpable liver and spleen, as so many tropical diseases are associated with an enlargement of these viscera.

You will therefore forgive me if I start my talk by reminding you about the correct method of palpating the liver. The busy practitioner is apt to abruptly dig his fingers into a patient's abdomen just below the right costal margin and thereby often miss an enlarged liver. One should invariably start gently in the right iliac foca with the palm slightly turned towards the diaphragm and move the hand towards the liver step by step, asking the patient to take a deep breath. It may be necessary to flex the legs in some patients to prevent the abdomen being kept rigid. By such careful palpation the liver, if it is at all enlarged, can be felt to ride over the palpating palm and tenderness can also be readily detected.

The causes of enlargement of the liver are

A. *Congestion.*

B. *Infections.*

1. Via hepatic artery.
   - (*a*) Spirochcto-icterohaemorrhagic; syphilis.
   - (*b*) Kala-azar ; Malaria.
   - (*c*) Typhoid ; Pneumonia.
   - (*d*) Infective hepatitis ; Yellow fever.
2. Portal infections.
   - (*a*) Amoebic.
   - (*b*) Suppurative Pyelephlebitis.
   - (*c*) Hydatid.

C. *Toxic Necrosis.*

1. Drugs–As.
2. Via portal vein–formerly cirrhosis of the liver and splenic anaemia used to be included under this heading ; their aetiology is now considered to be totally different.

D. *Metabolic Derangements* - pregnancy, diabetes, thyrotoxicosis,

E. *Nutritional* – Cirrhosis of the liver; massive necrosis.

F. *Blood Diseases* – anaemias, leukaemias lymphomas; Banti's disease.

G. *Carcinoma*

1. Secondary. 2. Primary.

We shall now consider each one of these conditions briefly with an extra emphasis on the more common conditions.

A. Congestion

The liver becoming enlarged, palpable and sometimes tender in the course of congestive heart failure is well recognised. But every now and then a patient with early congestive failure complains of a feeling of heaviness in the upper abdomen, a distaste for food, nausea and other dyspeptic symptoms rather than of breathlessness on exertion or swelling round about the ankle at the end of the day's work. In such an event the patient is treated symptomatically for the dyspepsia without the true nature of the illness being recognised. I have seen this happen more often with hypertensive congestive failure than with congestive failure complicating valvular disease.

Under this heading one should also remember the state of the liver in chronic constrictive pericarditis which is a late sequel of a tuberculous pericardial effusion. In every patient with apparent congestive heart-failure who does not respond in the usual way to the measures directed against congestive failure, the possibility of constrictive pericarditis being the cause of the cardiac distress, the enlargement of the liver and the ascites should be borne in mind. Screening and a careful history taking will help to decide the diagnosis. More cases of tuberculous pericarditis have been reported on in Indian medical literature during the last decade than in any previous decade. Many of the patients diagnosed in the acute stage of the illness have shown dramatic improvement with the specific antimicrobial drugs but it remains to be seen how many of them may report again with the clinical features of constrictive pericarditis.

B. 1 (b) Kala-azar and Malaria.

I would like to say a word with regard to the liver in Kala-azar and in malaria. Generally speaking, the liver enlargement in malaria is much less than the splenic enlargement whereas in Kala-azar the liver usually tries to keep pace with the spleen and occasionally, even overtakes it. This relative size between the spleen and the liver is an important consideration in differential diagnosis.

B. 1 (d) Virus Hepatitis

The most common types of acute hepatitis are the infectious hepatitis produced by virus A and homologus serum jauntice produced by virus B. While virus A is transmitted by the foecal-oral route, virus B is always transmitted parenterally through contaminated needles.

Virus A hepatitis is common under the age of 20 whereas virus B hepatitis occurs more often in middle age when apparently there is a greater exposure to blood and blood products.

A person may be a carrier of virus B for as long as five years even though he is clinically well. The incubation period of virus A hepatitis is about four weeks whereas that of virus B hepatitis is about three months. The diagnosis between virus A and virus B hepatitis is extremely difficult. Even if a liver biopsy is obtained, there is no correlation between the histologic picture and the type of virus producing the hepatitis and liver biopsy is not without risk in a patient with jaundice. On the whole, homologous serum jaundice is more

severe and more prolonged than virus A hepatitis.

I do not propose to go into the typical clinical features of infective hepatitis to this audience of post-graduates. But I would like to mention that when a child or young adult is seen with a fever with no localising signs, the possibility of virus hepatitis should always be remembered and the urine and conjunctiva seen during the daily visits. Otherwise the diagnosis is frequently made earlier by the grandmother much to the chagrin of the doctor.

Another point to remember is that the severity of the jaundice may vary within very wide limits. In fact, it is true of all virus infections that the range of severity is much wider than in bacterial infections. It is believed that in about 20% of adults and 90% of infants the disease may be an-icteric and therefore not diagnosed properly. While the average case may show a reduction in the degree of jaundice in two weeks' time, in severe cases the jaundice progresses consistently and causes the death of the patient in cholemia. Fortunately, this is said to happen in less than one per two thousand subjects. It is therefore important that every case of virus hepatitis should be treated with respect and should be kept under observation until complete cure is effected.

It should be the routine to get the icterus index done when the patient is seen first and again at weekly intervals till the return to normal values. By this simple procedure one can obtain timely warning about the uncommon serious case.

Laboratory tests :

In the early diagnosis of the disease, determination of the uro-bilinogen in the urine is more important than that of bilirubin. I feel that testing for this with Ehrlich's diazo reagent should be done much more frequently in clinical practice. Apart from ensuring the early diagnosis of the overt case, it will also help to provide data regarding the incidence of occult cases.

Serum iron which is about 130 micrograms per 100 ml. of serum shows a distinct rise in acute viral hepatitis to about 300 micrograms. In obstructive jaundice and jaundice following cirrhosis, it is only slightly elevated.

Trans-aminase can rise from the normal 22 units per ml. to several thousand units per ml. in acute hepatitis. In obstructive jaundice the rise is only slight.

While these examinations may not be available in many places and may not be necessary in most cases, they are extremely useful in the real problem case.

Treatment.

I often see patients, particularly doctors, remaining ambulant even though they are suffering from jaundice. I think bed-rest should be complete until the illness is fully recovered from, which usually takes about four weeks.

Diet. I have seen patients who have been put on a very meagre diet consisting only of barley-water, glucose and orange juice for several days. A patient with viral hepatitis should, on the other hand, be permitted to eat well-rice, dhal, butter-milk and orange juice with sugar,—ensuring an extra supplement of proteins in the form of the aminoacid preparations now plentifully available on the market. There is no objection to milk because of its fat content. With a diet adequate in proteins and rich in calories, there is no special indication for individual lipotropic factors such as choline or methionine. When the patient is able to eat well and retain the food, there is no indication for intravenous glucose infusions.

ACTH and Cortico-steroids are not recommended in the routine treatment of hepatitis of average severity. Their use is to be considered only under the following circumstances :

(i) patients with disease of such severity that hepatic coma is feared ;

(ii) patients with severe disease, intractable vomiting and with serum bilirubin in excess of 15 mgms. per 100 cc. ;

(iii) patients with cholangiolitic hepatitis. A reduction in the bilirubin level is undoubtedly brought about but the frequency of relapses is very much more in cases treated with ACTH or cortisone.

Saline purgatives are not necessary as a routine.

Antibiotics - have no specific action on the virus and are not indicated in the average case. If there is any fear of impending hepatic failure, tetracycline is used for reasons explained later.

Viral Hepatitis and Diabetes.

The incidence of enlargement of the liver and hepatic dysfunction has been found to be significantly higher in poorly controlled diabetics and the control of diabetes has resulted in improvement of hepatic function. Every now and then we see cases of viral hepatitis in known diabetics. Perhaps the infection is with virus B on account of the greater opportunity for infection of patients in diabetic clinics by improperly sterilized needles. It is, however, fortunate that viral hepatitis does not resemble bacterial infections in having a pronounced bad effect on the diabetes, but the combination of the two diseases undoubtedly leads to certain problems in the management of the patient. There should be no restriction in the caloric intake as long as the hepatitis is present. With the feeding programme described under the treatment of viral hepatitis, nhe diabetic subject will undoubtedly have the need for more insulin compared to his requirements before he got the hepatitis.

Virus Hepatitis-prevention.

The preventive methods applicable to all "enteric" infections will apply to this disease also.

Gammaglobulin in a dose of 0·02 ml. per pound of body weight injected intramuscularly has been widely used in Philadelphia and other places, especially, in the presence of epidemics. It may not actually prevent the disease but it undoubtedly reduces the severity.

Chlorpromazine cholangitis

The use of chlorpromazine (largactil) has resulted in jaundice in 1·27% of 8127 cases reported on by different workers and compiled by Spellberg. Even as small a dose as 50 mgms. has been followed by severe and longstanding jaundice. Apparently, it is a manifestation of idiosyncracy rather than one of toxicity.

Chlorpromazine hepatitis closely resembles virus hepatitis in its symptomatology. Pruritus is more marked and is present in a much larger percentage of cases compared to virus hepatitis. The elevation of alkaline phosphatase is pronounced. Chlorpromazine should not be used indiscriminately and is best avoided in patients with known gallbladder disease.

B 1 (d) Yellow Fever

It is not proposed to discuss the clinical features of Yellow Fever which one hopes will be kept out of India forever thanks to the vigilance of the Government of India. However, the danger of the disease entering the country is still quite real in view of the rapid air-travel between Africa and India and the prevalence of the Aedes Agypti, the insect vector of the virus.

B 2 (a) Amoebiasis.

Amoebiasis of the liver is, to my mind, the commonest cause of an enlarged liver in South India. Actual abscess formation is fortunately less common today compared to what it was about 15 years back. It is in no small measure due to the introduction of prohibition. While amoebic hepatitis has no relation to alcoholism, there is no doubt that alcoholism does predispose to abscess formation in the liver.

The picture of amoebic hepatitis is so familiar to all of us that there is no need to describe it at any length. I would only emphasize a definite history of amoebic dysentery, particularly with laboratory support to the diagnosis of the exact nature of the dysentery, is not to be expected in every case. Many patients complain of recurring attacks of diarrhoea and deny any previous episode of dysentery. The motion may not always reveal the presence of the cysts of E. histolytica as it undoubtedly requires experience and care to demonstrate the presence of the cysts. Even sigmoidoscopy may not show the presence of any active ulcers.

A complaint of heaviness in the region of the right costal margin and sometimes very severe pain in that area or over the base of the right lung or pain about the right shoulder are very suggestive symptoms which should always call for a screening of the chest to note the mobility of the right dome of the diaphragm.

Pulmonary amoebiasis should always be considered in the presence of any lung pathology over the right base associated with an enlarged and tender liver. It is not proposed to discuss the various manifestations of extra hepatitic amoebiasis.

I feel that to-day one can in most cases manage the treatment of amoebic hepatitis without emetine. The average South Indian patient, particularly of the hospital class, suffers from clear malnutrition and shows a low blood pressure. If we could avoid the use of emetine in him, it is certainly an advantage. Chloroquine di-phosphate is a real boon to us in the tropics. Its action in hepatitic amoebiasis is as effective as that of emetine and thedrug is absolutely safe. The symptoms of giddiness, nausea, anorexia, and blurred vision so often complained of can be ignored as they invariably pass off as soon as the drug is stopped and signify no permanent harm to the patient. During the last three years, I have depended entirely on chloroquine and never had occasion to regret it.

B 2 (c) Hydatid.

Hydatid is more commonly met with in district hospitals than in the City hospitals. The liver is one of the commonest sites and therefore in any obscure painless enlargement of the liver without jaundice, the possibility of hydatid should be remembered. While working at Madurai I had a lady, aged 30 years, whose leading complaint was a fullness in the right side of the abdomen. She had a liver extending to four fingers below the costal margin with no other symptom. Laparotomy revealed a large hydatid on the under surface of the liver stretching the liver over it.

C 1. Drugs.

A large number of drugs and poisons introduced into the body in different ways may produce a toxic necrosis of the liver. Of these, special mention should be made of arsenic in view of the rather liberal use of the drug in South India. I have seen several patients who have had more than one course of arsenic by injections and who have continued taking patent anti-asthmatic mixtures containing potassium arsenite for unduly long periods. Some of them have had all the evidences of chronic arsenical poisoning including a large liver.

E. Cirrhosis of the Liver.

Zimmerman has classified the types of alcoholic cirrhosis according to the stages in the development of the disease.

Type I, Simple Steatosis, shows mild to moderate fatty metamorphosis and is the earliest phase in the development of Laennec's cirrhosis. Clinically, the symptoms are mild and are largely those of alcoholic gastritis. There is hepatomegaly and spider nevi only in 5% of subjects. Of the various tests for impaired liver function, sulfobromophthalein excretion test alone may be abnormal in over 50% of subjects. The other tests show abnormality only in 10 to 15% of subjects.

Type II, Steato–cirrhosis, has been considered to be intermediary between simple steatosis and cirrhosis. The retention of sulfobromophthalein is of a higher degree and other tests such as thymol turbidity, cephalin flocculation and alteration of albumin globulin ratio are also abnormal in over 70% of cases. The symptoms such as epigastric discomfort, flatulence, nausea, are more prominent. Enlargement of the liver is invariable and evidences of portal hypertension such as prominent veins on the abdominal wall and splenomegaly are common. Gastro–intestinal bleeding may occur.

Type III, Cirrhosis, shows microscopically architectural distortion and strands of fibrous tissue with little or no fatty metamorphosis. The patient is usually older and the classical symptoms and signs are obvious. There is gross abnormality of all the liver function tests in 95% of the patients.

He has also postulated a variant of Type II which he has named steatonecrosis in which the metabolic failure of the liver precedes the manifestations of fibrosis. In this type jaundice and death in hepatic coma are commonly met with.

It is important to remember that the so called alcoholic cirrhosis is seen in its entire picture, histological and clinical, in patients who have never touched a drop of alcohol. It is believed that in them the nutritional deficiency culminating in cirrhosis is brought about by just malnutrition whereas in alcoholic subjects it is secondary to the alcoholic gastritis. Patek has noted that the incidence of cirrhosis among alcoholic patients is 8% as contrasted with the incidence of 1·2% in an abstinent population.

Dietary massive necrosis of the liver presents a puzzling problem in that in the clinic there is no equivalent of the experimentally produced state. In man while acute necrosis of the liver may follow infections and toxic insults, it does not appear to follow even severe dietary deficiency.

On the other hand, dietary factors play an undoubted role in the production of the various stages of cirrhosis. The lack of lipotropic factors, in particular of choline and its precursors, methionine and betaine leads to fatty infiltration and progressively to diffuse fibrosis and the terminal changes of cirrhosis. The lipotropic activity of protein is determined not only by its methionine content but also by the other aminoacids present and their relative balance.

Considering the millions of persons in our country subsisting on totally unbalanced diets lacking in the amino–acids and vitamins, the surprise is that there are so few cases of cirrhosis. It is therefore certain that dietary factors alone cannot determine the incidence of the disease. Infections like malaria, amoebiasis and virus hepatitis and the extent of damage they may cause to the liver cells must play an important role in the production of cirrhosis.

A normal liver synthesizes an average of 18 gms. of albumin daily. Approxi-

mately 40% of the total body albumin is found in the plasma, the remainder being extra-vascular. In most patients with cirrhosis and ascites, the albumin which is synthesized amounts to about half that which is normally produced.

In normal subjects, plasma gamma globulin determined by electrophoresis is about 0·85 gms. per 100 cc. The mean value in patients with typical Laennec's cirrhosis and ascites was found to be 2·3 gms. per 100 cc. Compared to normal subjects, in patients with cirrhosis the most striking abnormality in the metabolism of gamma-globulin was a greatly increased daily turnover.

Prothrombin activity in the plasma of patients with Laennec's cirrhosis averages 50% of normal. The fall in prothrombin activity in cirrhosis approximates the deficiency in plasma albumin.

A full discussion of the numerous liver function tests will be out of place in a clinical talk such as this. It has already been pointed out that the sulphobromophthalain excretion test is the earliest to show abnormality with impaired liver function.

While the laboratory does undoubtedly help in the diagnosis of most cases, occasionally a patient with cirrhosis may have normal hepatic function as determined by the usual tests. I would like to recall the case of C – who clinically had typical cirrhosis. He was a teetotaller. In 1951 he had such severe ascites that it had to be tapped thrice at intervals. But he improved later so well that he was able to continue in an active job which involved considerable touring. Early in 1957 the liver function tests which had been repeated several times before continued to show a very good functional efficiency. In March 1957 he died of a brisk gastric haemorrhage.

Ascites

The important causes of ascites are

1. Portal hypertension ;

2. Hypo – albuminemia, producing lowered osmotic activity of the plasma ;

3. Increased anti-diuretic hormone, resulting in water retention ;

4. Extravasation of hepatic lymph and

5. Retention of Sodium.

The restriction of sodium is usually effective in controlling ascites. The daily intake should be as low as 200 to 500 mgms. initially and can be increased gradually as the condition improves.

Paracentasis should be avoided as long as possible in view of the reported danger of precipitating hepatic coma. But where the distension is marked and produces respiratory distress, there is no alternative.

Mercurial diuretics may have to be employed if sodium restriction alone does not produce the desired results. Ammonium chloride usually prescribed before mersalyl in cases of cardiac oedema should not be employed in cirrhosis because of the role of ammonium ion in precipitating hepatic coma. Calcium chloride in a daily dose of 10 gms. has been employed as a substitute which may be continued throughout the period of mercury administration.

Diamox is also to be avoided in patients with serious liver damage.

Haemorrhage

Gastro-intestinal bleeding in a patient with known cirrhosis may sometimes be due to an associated peptic ulcer. In fact, some authorities believe that this is so in nearly 20% of cases. The demonstration of oesophageal varices is often difficult even with special radiological techniques.

The Sengstaken tube is employed to control the bleeding. It has an upper sausage-shaped balloon which compresses the oesophageal varices and a lower spherical balloon which presses firmly against the cardia. Blood transfusion is undoubtedly a life-saving measure and should be used with all the usual precautions.

There is a greater danger of the patient lapsing into a state of hepatic coma after an episode of severe bleeding because of the hypoxia of the liver leading to decreased hepatic function and the increased absorption of ammonia from the blood in the intestines. All necessary steps such as glucose infusions and the restriction of proteins to prevent neurological complications, should be taken.

Hepatic Coma

The role of elevated ammonia levels in the serum in the production of hepatic coma has been established. Increased protein intake may be the factor serving to increase the source of ammonia production. Therefore in any patient with impending hepatic coma, the protein intake should be severely cut.

Studies with radioactive carbon have established the role of intestinal bacteria in ammonia production. Drugs of the tetracycline group are considered most useful in keeping down intestinal bacterial activity. It is seldom necessary to continue the anti-biotic for more than a week.

Sodium Glutamate has been shown to be capable of lowering blood ammonia levels. Sodium Glutamate should be sterilized by means of bacterial filters and not by boiling or autoclaving and given in a drip of 5% glucose.

Potassium and phosphate should be given according to the needs of the patient determined by their concentration in the plasma.

In the comatose patient attention to tracheal suction and maintenance of a clear air-way are important.

Reports on ACTH and cortisone in cirrhosis have been variable. They are not to be used in the average case. Actually, portal thrombosis, hematemesis and flaring up of obscure infections elsewhere in the body have been reported as complications of cortisone therapy. However, they are used in cases showing biliary retention and as a desperate measure in cases which are going down.

Cirrhosis of the Liver – Treatment

Certain preparations of whole liver extract such as Intraheptal and Riposan are decidedly useful in the early stage of the disease. How exactly these preparations act in producing a distinct clinical improvement is not known. When the injections – intravenous in the case of Intraheptal and intramuscular in the case of Riposan – are maintained indefinitely, the progress of the disease is kept under check even in some patients who have had one or two abdominal tappings.

Surgical treatment.

Portal systemic venous shunts have been employed in an increasing measure during the last decade to reduce portal pressure and thereby decrease the incidence of haemorrhage.

Portacaval shunt produces the most effective de-compression and is the operation preferred in most cases with cirrhosis of the liver.

Spleno-renal shunt with splenectomy is more conservative and can be used for both intra-hepatic and extra-hepatic blocks.

Patients with severe liver damage are unsuitable for these surgical procedures.

Patients must be prepared pre-operatively with medical measures directed to improve the liver function. The brom-sulphathalein retention should be reduced to 30% if possible. Ascites should be relieved before operation.

Kwashiorkor.

This is a condition seen frequently in South Indian children. It has been shown that while total caloric starvation may lead to general emaciation in which the liver may show a diffuse a trophy, the disproportionately low intake of protein, especially protein of high biological value, with normal or almost normal caloric intake results in kwashiorkor. The liver undergoes fatty infiltration, serum albumin is reduced, generalized oedema appears and the child is prone to intercurrent infections. It has also been pointed out that true cirrhosis is uncommon following kwashiorkor possibly because either the child dies prematurely or the condition is cured by adequate nutritional treatment.

F. Blood Diseases.

It is not proposed to go into a full discussion of the various blood diseases that are associated with an enlargement of the liver. While the diagnosis of the anaemias and leukaemias may be comparatively easy if one takes the pains to carry out proper and thorough investigations, the diagnosis of the lymphomas is often very difficult. The enlargement of the liver and spleen in the course of a lymphosarcoma in a doctor was put down to kala-azar on totally inadequate grounds. A girl of 10 with enlargement of the liver and jaundice was treated as infective hepatitis for an unduly long time while she was really suffering from Hodgkin's disease. The lymphomas should always be borne in mind in dealing with many obscure diagnostic problems.

Banti's disease is now regarded as the continuing stage of erythroblastosis foetalis in survivors. The etiological factor is rhesus incompatibility in the parents and the action of Rh agglutinins on the foetus.

G 1 Secondary.

The liver is the site of secondaries, early when the primary is in the region of the drainage of the portal vein and somewhat later when the malignant cells travel first through the lungs and then reach the liver. Although one generally thinks of cancer only after the age of 40, it is by no means unknown in younger subjects. Quite recently, I saw a young woman of 22 with a hard painful liver and slight jaundice. Investigations revealed a primary growth in the lower third of the oesophagus but the patient had never complained of dysphagia.

The hepatic radio activity survey has been used to obtain a reliable indication of the presence of liver secondaries. It has proved more dependable than even biopsies.

G 2. Primary Hepatoma of the liver

This condition is more common in countries where malnutrition is widely prevalent, for example, Africa and perhaps, South India. In addition to protein malnutrition, the increased deposition of iron in the liver in siderosis appears to be a cause of primary carcinoma.

Partial hepatectomy, during which more than 50% of the liver tissue has been removed, has been employed in the treatment of primary carcinoma.

RECENT ADVANCES IN EAR SURGERY

by

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TWENTY years ago, when I was a house surgeon in the E. N. T. Dept. of the Govt. Genl. Hospital the only type of Surgery which was done for the ear was the Mastoid operation. The operation, as we all know, is for the infections of the ear and mastoid. Even this surgical procedure was not thorough since we were mighty frightened of all the important anatomical structures in the field of operation: the sinus, the facial nerve, the middle cranial fossa and the semi circular canals. We would just lay open the antrum, curette some and not all of the mastoid air cells and close up the wound. Whenever a radical mastoidectomy was attempted we considered ourselves lucky if the patient did escape a facial paralysis post operatively. As for the hearing a further defect in hearing post operatively was invariably expected. In those days we never did any surgery to improve the function of the ear: 'hearing'. The only thing we ever did for those hard of hearing was to pass an eustachian catheter and blow air to open up the eustachian tubes.

But during the last two decades ear surgery has undergone rapid progress. We now have three surgical procedures for the ear and all of them are concerned with improving the functional aspect of the ear. Today I propose to give a brief outline of these three procedures.

1. Fenestration surgery of Lempert

2. Stapes mobilisation of Rosen

3. Tympanoplasty which may be called after the great german otologists Prof. Zollner and Prof. Wullstein.

The first two surgical procedures, Fenestration and stapes mobilisation are concerned with the improvement of hearing in one type of conductive deafness. This deafness is known by the name "Otosclerosis". Some years back, when I had the privilege of talking to the Trichy branch of the Medical Association I spoke about otosclerosis and the fenestration operation in detail. I also exhibited Lempert's film on the fenestration operation. Hence I do not propose to deal with this operation in detail. But for the benefit of those that were not there then I will speak briefly about otosclerosis and fenestration operation.

Otosclerosis is a type of middle ear conductive deafness which manifests itself during adolescence and is familial in nature. At least 60% of middle ear deafness is due to otosclerosis. The pathological process which is really in the nature of "Otosporosis" involves the bony capsule of the inner ear. It may be present anywhere in the bony capsule but 'the site of predeliction' for this pathological process is in the region of the "Oval window". Therefore with the spread of this pathological process in the region of the oval window there results some sort of ankylosis of the foot plate of the stapes to the oval window. Once this ankylosis takes place the ossicular chain which conducts the sound waves from the ear drum to the inner ear is interfered with resulting in partial deafness. The patients with otosclerosis have normal ear drums, normal middle ear and patent eustachian tubes. Since their sound perceiving mechanism, i. e, the organs of Corti the cochlea and the nerve of hearing is normal, they hear better when a tuning fork or a wrist watch is pressed over the mastoid bone. what is, their bone conduction is better than air conduction. Neither medicine nor the old method of eustachian

catheterisation will help in these cases. Therefore, a person suffering from otosclerosis can choose one of the two things to improve his hearing and they are :

1. An electrical hearing aid to magnify the sound as in spectacles for vision.

or 2. Undergo one of the two surgical procedures : the fenestration operation or stapes mobilisation.

Fenestration operation.

This operation consists in doing a radical mastoidectomy with partial exenteration of cells and secondly removing the incus and cutting the head of the malleus. The third stage consists in making a flap consisting of the ear drum and the skin lining the postero-superior aspect of the external auditory canal and is used for covering the floor of the mastoid cavity and the middle ear as also the newly created window. The fourth stage is the making of the new oval window which should be on the horizontal semi circular canal. This will be in place of the old oval window which is closed up by the ankylosis of the foot plate by the pathology. Thus in a fenestrated ear the new oval window and the round window which is already present function for the conduction of sound waves from the exterior to the inner ear.

The entire operation takes about two to three hours and the patient has to stay for about 12 days in the hospital after which another six weeks of cleaning and dressing is needed. For two or three weeks after the operation the patient has giddiness and nausea.

Stapes mobilisation.

This operation is comparatively a very simple one. The steps of the operation are :

1. Incise the skin of the external ear canal for about half an inch from the drum margin from the 12 O'clock to 6 O'clock position and lift it up from the bony canal up to the ear drum.

2. Lift the ear drum also from the Sulcus Annulus posteriorly from the 12 O'clock to 6 O'clock position. Then throw the ear drum forward over the remaining anterior half of the drum thus exposing the posterior half of the middle ear space. Now one can visualize the tip of the long process of the incus above and the round window niche below.

3. With a delicate bone curette we curette the lower bony margin between the 12 O'clock and 9 O'clock position till the long process of the incus, incudostapedial joint, the crura of the food plate of the stapes are visualized.

4. We then try to mobilize the foot plate of the stapes either by trying to move the stapes at its neck or at the margin of the foot plate. We succeed in mobilizing the stapes at the oval window by breaking off the ankylosis in certain number of cases. The patient immediately hears better. In at least 50% to 60% of the cases we fail to mobilize the foot plate because the ankylosis is too firm and bony. In these cases the crura, often gives way. Even in those where mobilization has been effected there is no guarantee that the stapes will remain mobile for a long time. The pathology is not removed and at least 50% of the cases where we succeed in mobilising, ankylosis recurs in 2 or 3 weeks. So the percentage of success is only about 20 or 25%.

The advantages of this operation over the fenestration operation are :

1. The operation is simple and takes only 20 or 30 minutes.

2. There is no post operative giddiness or nausea.

3. The operated ear heals up completely in a week and the patient can resume his work in a few days : a week or ten days.

4. If this technique fails we can still do, the fenestration operation after 2 or 3 months.

5. Since the operation and the post operative convalescence are so short it can be tried even in doubtful cases : cases having mixed deafness and cases where we are not sure of success, we can try this without much inconvenience to the patient.

Tympanoplasty.

This recent and new operation, and as the name suggests consists in plastic repair of the damaged or perforated ear drums. Therefore this is meant not for persons having defective hearing because of otosclerosis but for those who have defective hearing because of chronic middle ear infection where the ear drum and the ossicular chain is damaged. Till recently we had no method of treating patients with defective hearing due to these causes except recommending a hearing aid. Thanks to the development of micro surgery by Dr. Lempert and others for otosclerosis and thanks to the anti biotics for the control of infections we have reached another mile stone in otology, viz. tympanoplasty. There are several tasks to be achieved in this surgery.

1. Chronic infection of the middle ear and antrum must be eliminated and the ear must be dry before undertaking the operation.

2. Must build a new air filled closed tympanic cavity which must be properly ventillated through the eustachian tubes.

3. A new conducting system for sound through the middle ear to the chochlea must be constructed. This conducting system must be modified according to the degree of damage in the middle ear.

In selecting a case for this procedure the following clinical factors which influence the operation must be borne in mind.

1. *Quality of the inner ear.* If there is a preponderance of nerve deafness the result of the operation may not be very good.

2. *Function of the eustachian tube.* Many, if not all cases of middle ear infection have granulation tissue and pathology in the eustachian opening at the middle ear space.

3. Hydrodynamics of inner ear depending on the round window and oval window conditions.

4. Quality of the mucosa of the middle ear.

5. Quality of the remaining ear drum.

6. Condition of the ossicles, whether partially destroyed, absent, etc.,

Depending on these various factors the surgeon has to decide on the type of operation he is to do. There are about 5 or 6 types of operations which we can adopt. In some both "sound protection for the round window" and "sound pressure transformation on the oval window" can be maintained. But in those where the malleus and the incus have to be sacrificed or are absent sound protection at the round window alone can be preserved without the sound pressure transformation at the oval window.

The operation, like the fenestration is an elaborate and time-consuming one and the success depends on the way in which the infection is eliminated before applying the skin graft. The skin graft for repair of the damaged drum is taken from the thigh or from the arm or from behind the pinna. We usually use a thin full thickness skin graft.

I do not want to bother you with the technical details of the operation. I only want you to know that this new operation is for those cases where chronic middle ear infection is responsible for loss of hearing, or where the drum is partially damaged which leads to loss of hearing.

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CORTICOSTEROIDS

by

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SOME of us might still recall the bold headlines in 1949 when the effects of the " wonder drug " on the cripples of rheumatoid arthritis flashed out in the newspapers all over the world.

Kendall's compound E – Cortisone–was thought by many as panacea in most of the diseases. Hence, the Noble Prize Winner, rightly struck the note of cautious optimism. Inspite of tremendous amount of fruitful clinical and experimental work, there are divergent views to justify their uses and their mode of action. There is, no doubt, that these drugs if used properly, are a very valuable addition to the armamentarium of a medical man. Because of the prompt and salutary effect, the clinician might be tempted to use this drug in conditions where it may not be justified. It could be a dangerous drug.

The growth of voluminous literature regarding the drugs makes neither possible nor wise to go into details. This presentation makes no claim to discuss all the aspects.

Mode of Action

Under ordinary conditions, hydrocortisone is always produced in the body by the stimulation of the adrenal cortex in varying amounts through the medium of corticotrophin or A. C. T. H. secreted by the anterior pituitary gland. Apart from variations in the amount secreted in the different hours of the day and night, such stress as mental and physical strain, trauma, infections, surgical operations and anaesthesia, provoke cortical secretions.

Patients with adrenal insufficiency react to all forms of stress badly which only means that these cortical harmones are needed under such circumstances.

Active secretions are glucocorticoids, *i.e.*, hydrocortisone, mineralo-corticoids including aldosterone, and other sex harmones. The exact mode of action is still conjectural and it must be realised that these drugs are not curative. Glucocorticoids have an anti-inflammatory, anti-rheumatic, anti-toxic and anti-allergic properties. They reduce capillary permeability, suppress the formation of granulation and fibroblastic tissue and depress the cellular oedema. Their action is mostly suppressive but may also be used to supplement cortical dificiency. They do not affect the noxious aetiological agents directly but modify the tissue reaction. They have a sinister tendency to aggravate the infections of any description but suppress fever and impart a sense of well-being. They reduce the formation of anti-bodies and diminish phagocytosis.

In most of the diseases, their use, has been empirical. It is justifiable that a correct diagnosis is arrived at before the institution of these drugs. They are powerful agents but at the same time are liable to produce serious side-effects.

Preparations, Method of Administrations, Dosage :

PREDNISONE and PREDNISOLONE—have replaced cortisone and hydrocortisone.

These synthetic preparations are also available for parenteral use, being five times more potent. There is, however, nothing to choose between these two newer preparations. There are other new preparations in the market which have not been widely used i.e., LEDERCORT (triamcinolone) and are said to produce less side-effects.

In extreme emergencies, hydrocortisone - 100 mg. - intravenously with saline or glucose drip is used. Intramuscular preparation of cortisone is advocated in vomiting or coma. It must be remembered that the absorption occurs more rapidly by oral route than when given by intramuscular injection. Hydrocortisone can also be given intraarticular, and locally in the muscles and tendons. Eye drops, nasal drops and ointments are also employed. In the administration of these drugs gradual reduction by stages should be undertaken ("tapering off"). A sudden withdrawal may precipitate a "rebound" phenomenon. Except for adrenal deficiency, where a dose of 5 to 10 mgs. of Prednisolone may be sufficient, the dosage in systemic diseases should be high (aggressive dose) to begin with and once a therapeutic effect has been achieved, it should be weaned off gradually. Severe complications can occur with big doses especially if administered for a prolonged period. Fine hydrocortisone acetate powder (15 mg. of hydrocortisone with 75 mg. of lactose) from an inhalar given twice or three times a day in acute recurrent bronchial asthma with symptom free intervals has shown good response and is cheeper than these steroids given orally. However, higher dosage of corticosteroids will depress the secretion of A. C. T. H. from the anterior pituitary gland, thereby causing hypofunction of the adrenals. Under such circumstances injections of A. C. T. H. 20 units, 3 or 4 times, may be used to stimulate adrenal cortex. It is not given as a supplementary therapy in cortical deficiency.

Its place in therapy has been largely taken up by corticosteroids. Potassium (KCl or Citrate) should be given if these drugs are administered in big amounts.

Side-effects

Adverse effects may vary from mild dyspepsia to death. There should be, however, no cause of alarm, remembering that all drugs can be potentially dangerous, nor is it that in every case of steroid therapy side-effects supervene. Clinical efficacy must always outweigh the side-effects. Their severity and frequency depend upon the length and the intensity of the dosage used.

Gastrointestinal: Prednisone has the reputation of producing dyspepsia, increased gastric acidity, activation of a peptic ulcer with or without haemorrhage and perforation. Wisdom lies in eleciting such a history beforehand, especially when peptic ulcer happens to be so common in this part of the world. There is, however, no contraindication to treatment even if there is history of pre-existing peptic ulcer. To avoid this complication, corticosteroids should be given after meals or with magnesium trisilicate or aluminium hydroxide.

Gain in weight: It is the result of increased appetite and rarely due to the retention of salt and water. Our patients who are generally underweight, an increase in weight is always beneficial. However, in an obese patient low caloric diet is indicated.

Diabetes mellitus: Mild hyperglycaemia with glycosuria occurs in a few patients and in some it may persist after the termination of the treatment.

Diabetes mellitus does not contraindicate their use if the necessity of these drugs has been considered important. Insulin requirement has, of course, to be stepped up.

Tuberculosis: Tuberculosis happens to be another complication and should always be kept in mind when these drugs are given for a prolonged period.

Cushing's syndrome: Rounding of facial contour occurs with prolonged therapy, sometimes accompanies hirsuitism and acne vulgaris. Bruising of the skin, purple striae and osteoporosis arise due to weakening of the connective tissue. Spontaneous fractures and collapse vertebrae especially in old persons and chronic cases of rheumatoid arthritis have been recorded. A severe pain in the back should give warning of a bony lesion. A high protein diet with potassium is advocated in all these cases with prolonged treatment.

Hypertension occasionally complicates the therapy. Amenorrhoea or oligomenorrhoea may occur with or without hirsuitism and acne vulgaris as a result of androgenic action of those drugs.

Electrolyte imbalance: Sodium retention and potassium depletion used to be common during the administration of cortisone and A. C. T. H.; with the advent of newer drugs, this danger is hardly confronted with. With bigger doses it is better to restrict the salt and administer potassium (Kcl-1 G t. d. s.)

Psychological and neurological disturbances: Euphoria had been said to occur with steroid therapy. Apart from a sense of well-being and a feeling of good health, we have not noticed any signs of excessive hilarity. Moods of depression arise after sudden withdrawal, if used in large amounts. Convulsions had been reported to occur.

Blood coagulation: Thrombo-embolic episodes are liable to occur and may need larger doses of anti-coagulants to maintain the plasma prothrombin activity.

Hazards of major surgery: Cases who have had corticosteroids are considered potentially weak to withstand all major stress, including major surgery. At the time of such operations, they are given supportive therapy with these drugs to avoid severe hypotension and shock.

Withdrawal symptoms: Due to the disuse atrophy of the adrenal cortex with high doses of corticosteroids a sudden withdrawal causes nausea, vomiting, restlessness, joint pains and aggravation of symptoms. This therapy, should, therefore, be tailed off or injections of A. C. T. H. should be given for few days to stimulate the suprarenal cortex as already mentioned.

A few cases of rheumatoid arthritis with prolonged therapy have been known to develop periarteritis nodosa.

Use of Corticosteroids

(a) Suppressive therapy.
(b) Replacement therapy.
(c) Investigations.
(d) Research.

(a) **Suppressive therapy:**

(1) *Nephrotic syndrome:* There are various causes of nephrotic syndrome but the administration of cortisone or prednisone has been instituted in glomerulo nephritis. The dosage should be 60 mg. of prednisolone daily for 1st week, 40 mg. for next week, 30 mg. for third week, 20 mg. for fourth week and then depending upon the response the amount should be gradually reduced. The course can be repeated. Best results are obtained with high initial doses. Failures are common with small doses. It should be remembered that children may need as much as adults. Patients with hypertension respond poorly. Second course may be given for relapses.

(2) *Bronchial asthma*: These drugs are reserved for severe intractable cases. In status asthmaticus, hydrocortisone – 100 mg. is given in a glucose drip at the rate of 25 to 50 mg. in the first half an hour. In severe cases 10 mg. of prednisone is given four times a day. A maintenance dose of 5 mg. 3 or 4 times a day can be given for a prolonged period. It is advisable to combine anti-biotics during their administration. Use of inhalation of hydrocortisone powder has been discussed already.

(3) *Rheumatoid arthritis*: An ideal case is the one in whom disease is limited to the soft tissues and does not involve bones and cartilages. But advanced cases have been given these drugs before the application of serial plasters. Patients suffering from spondylosis ankylopoetica have responded when they had proved resistant to other forms of treatment. In juvenile rheumatoid arthritis and Still's disease beneficial effect has been known to occur. Most of the cases respond to initial doses of 5 mg. of prednisone four times a day. It is better to combine it with aspirin-between 40 to 60 grains – daily.

It may be pointed out here that for a long-term therapy, there is little to choose between aspirin and corticosteroids in most of the cases. We have only advocated their use when large dose of aspirin have failed to show adequate response.

Most of our cases, who have had short-term therapy, have relapsed, though at the time of administration they had shown remarkable improvement. Whatever be the choice of drugs and the treatment, physiotherapy and orthopaedic procedures should not be given a minor place in the management of all these forms of arthritis. Cortisone or prednisone has hardly a place in the treatment of osteoarthritis.

(4) *Rheumatic fever and rheumatic carditis*: There is a difference of opinion as regards the effect of hormonal therapy in these conditions. Failures have mostly been noticed with smaller doses. These steroids should be combined with aspirin and given in the same doses as mentioned for nephrotic syndrome. Fever, tachycardia, E. S. R., electrocardiographic changes and other toxic effects improve in a short time. Pericardial effusion and friction rub sibside while murmurs disappear. In well controlled trials with this combination of aspirin and cortisone, very few obviously residual signs remain. No new manifestations of rheumatic fever develop during the steroid therapy which may occur with salicylates. Congestive heart failure recedes. It is better to supplement them with diuretics, digitalis and a salt free diet.

(5) *Infections*: In fulminating septicaemia associated with meningo coccal infection, Waterhouse-Friderichsen syndrome and other infections, i.e., pneumonia with circulatory shock, typhoid with chloremphenicol therapy, these drugs have proved life saving, the dosage being the same as that for Addisonian crisis

Hydrocortisone is given in a glucose-saline drip. Intravenous steroid therapy has a place in toxaemic shock. There are many case-reports where death has been prevented by the use of these drugs. In quite a number of cases, after thoracic surgery, severe intractable hypotension has occurred. Inspite of noradrenaline drip, the response has been disappointing. In some of our cases hydrocortisone has saved lives.

(6) *Liver diseases*: In few cases of infective hepatitis with prolonged duration, a relentless progression has been terminated by cortisone. Our series is a small one and enough time has not passed to make a final assessment. We

have had discouraging results in hepatic coma. It may be that our doses have been smaller. Some writers have advocated 1,000 mg. of cortisone per day.

(7) *Blood disease* : In acquired severe haemalytic anaemia, some cases of severe idiopathic thrombocytopenic purpura and agranulocytosis, these steroids have shown gratifying results, and are given in big doses. Acute leukaemias show good results but eventually and fatally.

(8) *Collagen diseases*: Periarteritis nodosa, lupus erythematosus with visceral manifestations, dermatomyositis and giant-cell arteritis (temporal arteritis), whatever may be their etiology, show modifications in their tissue reactions. Hypersensitivity reactions, allergic conditions and radiation, reaction the response is usually rapid and favourable.

(9) *Tuberculosis and tuberculous meningitis*: Because of anti-inflammatory properties and the ability to inhibit the formation of granulation and fibrous tissues, steroids make anti-tubercular treatment more effective and have reduced the impending hydrocephalus. They also diminish the allergic reaction which sometimes occurs with anti-tubercular drugs. Nevertheless, the treatment is only confined to severely ill patients.

Corticosteroids have also proved effective in mumps orchitis lepra reaction, acute encephalitis or encephalomyelitis, acute gout, malignant tumours of Islet's of Langerhans with metastases (because of its hyperglycaemic effect), Reiter's syndrome, pulmonary fibrosis, thyrotoxic crisis and sarcodosis with or without hypercalcaemia, reducing the side-reactions of nitrogen mustard in the treatment of Hodgkin's disease and other malignant conditions.

Corticosteroids have shown a good response in moderately severe cases of non-specific ulceretive colitis. Perforation, haemorrhage and pyoderma have complicated the therapy.

(b) **Replacement therapy :**

(1) *Adrenal insufficiency :* In Addison's disease or in patients with bilateral adrenalectomy for malignant hypertension,, carcinoma breast and prostate, 5 to 10 mg. of prednisolone is given daily in divided doses. In Addisonian crisis, 100 mg. of I. V. hydrocortisone with glucose saline drip should be given.

(2) *Adreno-genital syndrome* ; This may be due to an aberrant adrenal dysfunction rather than over-function. Failure of biosynthesis of corticosteroids occurs which fails to inhibit the secretion of A. C. T. H. from the pituitary gland. If it is present in utero, female pseudo-hermaphrodism occurs with enlarged clitoris, advanced skeletel bone age and persistent urogenital sinus. Any stress due to different aetiological causes can be lethal in infancy by producing Addisonian crisis. In a male child it can produce precotious puberty while in an adult woman signs of virilism with undeveloped breasts and failure to menstruate. In all the above conditions, 17 ketosteroids in the urine are raised to high figures.

The administration of corticosteroids inhibits the secretion of A. C. T. H. which fails to stimulate the adrenal cortex in return.

(3) **Anterior hypopituitarism** : This condition with or without other forms of therapy shows marked improvement with corticosteroids. Patients who have been hybernating or depressed, can be restored to relatively active life. The dose used is the same as that required for Addison's disease. In the event of any infection, however trivial, the treatment should be doubled or trebled.

Local Treatment : Hydrocortisone has been injected in the joints in rheumatoid arthritis, around the joints in the frozen shoulder or tendinitis. Eye drops and skin ointments of these drugs have given favourable results. In syphillitic interstitial keratitis since the use of hydrocortisone, the prognosis has been very favourable.

SOME PAEDIATRIC PROBLEMS

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CHOANAL ATRESIA.

Respiratory distress in the new born :

There are several causes for the respiratory distress in the new born. Choanal atresia is one of them and this is frequently missed and mistaken for congenital abnormalities of the tracheo-bronchial tree or tracheo-oesophageal fistula.

Choanal atresia : This is a rare congenital anomaly. This may be unilateral or bilateral. This may be complete or incomplete. It may be osseous or membranous or mixed. Many have osseous obstruction. 2 cases have been reported in male siblings in successive pregnancies.

Anterior choanal atresia and stenosis is rare. *Post choanal* atresia is occasionally seen. It is inherited as a *dominant* trait in some cases. Females are more affected than males, 2 : 1.

Clinical picture : The clinical picture of bilateral choanal atresia is one of 'Respiratory distress' in the neonatal period. This is exaggerated during feeding.

Case : A five day old infant was admitted because of frequent episodes of *respiratory distress and cyanosis.* Feeding caused choking. Hence tracheo-oesophageal fistula was considered at that time.

Physical examination *:* revealed a well developed infant with substernal retractions during inspiration (unless the mouth was open). At times, the infant became cyanotic, when the infant cried, the cyanosis was relieved. Attempts at feeding caused 'choking spells' for the infant could not nurse or breath at the same time. So there was gaging and coughing.

Tracheo-oesophageal fistula, was ruled out for a catheter could be easily passed into the stomach. Then it was suspected that this could be a case of posterior choanal atresia for no air exchange could be detected in either nostrils. A No. 8 French catheter could not be passed through either nostril into the pharynx. Inability to pass the catheter, does not establish a diagnosis for there may be *a pin point opening* which may be sufficient for nasal air exchange. Diagnosis was confirmed by holding a piece of cotton (or tissue paper) in front of each nostril to see if it vibrates during respiration. Finally lipiodol was instilled into each nostril and pictures taken in A. P. & Lat. views to see if there was any block.

X-rays : 1. X-ray showing probe in the nostril meeting the obstruction.

2. Lipiodol instillation showing the block.

Treatment : Surgical. This is a surgical emergency. Operation is done even on the 6th day of life. In about 90% obstruction is bony. Transpalatal approach favoured, because narrow nasal passages are easily abraded by transnasal instrumentation and adhesions develop.

PULMONARY HYALINE MEMBRANE DISEASE

Another cause of respiratory difficulty in the new born period is '*Atelectasis*' This may be primary or secondary. Primary atelectasis is often seen in a premature infant for there is failure of lung expansion. One of the causes of secondary atelectasis is pulmonary 'hyaline membrane disease' This is seen in

1. Premature infants especially under $4\frac{1}{2}$ lbs.

2. Babies delivered by caesarian section even after full term i.e., esp. of diabetic mothers.

3. In babies born of diabetic mothers.

4. Sometimes even in normal infants-esp., infants of mothers with placenta previa.

This condition is *not seen* in

1. Still births.

2. Infants dying within a hour.

Clinical features: The important clinical symptom is 'extreme respiratory distress'. There is difficulty in inspiration and this is accompanied by laboured respirations with chest retrations — suprasternal and lower thoracic with increasing cyanosis and respiratory grunt. Therc may be difficulty at resuscitation of birth. Some are born normal at birth. There is usually a brief 'good' period, lasting 1—3 hours and never over 12 hours. After this period, the infant shows respiratory distress with increasing dyspnoea and cyanosis. Physical examination reveals poor air exchange in spite of vigorous respiratory movements. They frequently die without complications in 24—48 hours. Infants dying later have complications like pneumonia, pnemothorax and pnemomediastinum. Some of these infants may survive if they are carried over the first 4 or 5 days of life. If the infant survives the recovery is complete.

Illustrative cases: Case—1: A baby was delivered by caesarian section after full term (non-diabetic). With great difficulty the baby was resuscitated. When the breathing was established, the colour and cry was good. Approximately 7 hours later, chest retrations were noticed in the lower intercostal spaces. The infant was placed in high humidity with oxygen 30%. Antibiotics were given. On physical examination there was diminished air entry in the chest. There was also slight cyanosis. The condition gradually became worse and the infant expired the next day.

At autopsy this was proved to be a case of pulmonary hyaline membrane disease.

Case — 2: I have seen another similar case in an infant born of a diabetic mother. This infant was also delivered by caesarian section. In this case, there was no need for resuscitation. The infant developed similar symptoms 7 hours after birth and expired in 48 hours. Infants of diabetic mothers may die of respiratory complications like hyaline membrane disease instead of hypoglycaemia.

Case — 3: This is a case which recovered (diagnosis by clinical grounds.) A white female infant aged 2 days was admitted because of chest retration and slight cyanosis. This infant was born full term — transverse presentation. The infant breathed spontaneously at birth. B. W.—9 lbs.—7½ oz. Because there was some cyanosis at birth, the infant was placed in oxygen for possible atelectasis.

Approximately 2 hours after birth, chest retractions were noted, and the infant had recurrent episodes of cyanosis. So oxygen and humidity were continued and in addition antibiotics — penicillin and streptomycin were given. Resp. rate was 90—100. Chest was hyperresonant with diminished air entry. Liver just palpable.

X-ray of the chest revealed generalised mottling throughout the lungs. Two days later breath sounds improved, colour remained good and respiration less laboured — respiration 30 — 40 mm. At this stage oxygen was discontinued but humidity continued. The infant

continued to progress well. She was discharged after 14 days stay in the hospital. Her weight at the time of discharge was 8 lbs. 12 oz.

X-ray lungs taken 10 days later after the 1st x-ray showed partial clearing. Three weeks later there was complete clearing.

Treatment :

Preventive :

1. Since this is seen in prematures, prolong the period of gestation
2. Caesarian section to be done only when there is real indication. When caesarian section is done, do gastric aspiration.

Treatment :

1. Oxygen in good concentration—30—40%.
2. High humidity—95 — 100%.
3. Penicillin (Procaine) 100,000 units B. D. and streptomysin (150 mgm per day) Provocative 100,000 unit B. I. D.
4. Do not feed when there is respiratory distress and poor sucking

— I. V. FLUIDS IF NEEDED.

Prognosis :

1. 50% recover. These have usually mild symptoms.
2. Infants who live for 72 hours—chances of recovery are better, unless some complications like pneumonia sets in.

A CASE OF CARCINOMA OF THE THYROID

Carcinoma of the Thyroid gland :

Though it can occur, this is a rare condition in children. A child of 8 years (girl) was admitted because of enlarged cervical glands and a nodule in the thyroid. This case proved to be one of adenocarcinoma of the thyroid with metastatis in the cervical glands, The lungs were clear. Hemithyroidectomy with removal of all cervical gland was carried out in this case.

Often the nodule is missed and such conditions are treated as tuberculosis of the cervical glands, Hodgkins etc. It is also a wise policy to feel the thyroid for any nodules in all masses in the neck.

Some cases of carcinoma of the thyroid occurred after x-ray treatment for enlarged thymus gland. But this was not seen in all cases of irradiation during infancy for enlarged thymus. But the risk is there.

A FOREIGN BODY IN THE BRONCHUS SIMULATING BRONCHIAL ASTHMA

When one hears wheezing in the chest, a diagnosis of bronchial asthma is usually made. But all that wheezes is not asthma. The following case illustrates how a foreign body in the bronchus can simulate bronchial asthma.

A 3 year old child was admitted into the hospital with history of (*a*) wheezing respiration (*b*) productive cough (*c*) foul smelling breath (*d*) intermittent chest pain.

All these symptoms were present (since the age of 1 yr) for 2 years. The wheezing was present throughout the year and it was most noticeable in the night. Mother also noticed that the child had some difficulty in swollowing for he took a long time to chew his food. He had been to many doctors since the age o one year and was treated with various medicines as a case of bronchial asthma without relief.

Physical examination revealed a well developed child with no distress. There was unpleasant odor in the breath. The chest was resonant with generalised expirator wheeze.

Mantoux test was negative.

X-ray of the chest – A. P. and Lat. views revealed a foreign body, a nail in the left main bronchus.

This was removed. It was a 1″ long nail which was rusty. Dysphagia was due to the pressure of the nail on mid oesophagus. No serious complications of foreign body like obstructive emphysema, atelectasis, bronchiectasis, pneumonia and abscess was seen in this case for there was only a small tissue reaction by the metal and the position was such that it failed to produce any bronchial obstruction.

A CASE OF HIRSCHSPRUNG'S DISEASE IN AN INFANT.

This is one of the rare causes of constipation in infancy and childhood, It probably occurs 1 in 20,000 – 30,000 live births. Boys are more affected than girls. The presenting symptoms are severe constipation and enlargement of the abdomen, due to dilatation and hypertrophy of the colon. This has a characteristic radiological picture with a dilated colon terminating in a ' narrow segment ' of a variable length comprising the sigmoid and rectum. In the narrow segment, the ganglian cells are absent. This condition is operable (recto-sigmoidectomy) and there is complete cure. This condition is genetically determined, and the mode of inheritence is not known. There is a familial incidence. When one child is affected in a family, there is probably 1 in 5 chances for and subsequent boy being affected. In this condition the development of the ganglian cells is interrupted in embryonic life.

Case: A male infant (1st child) 4 months old, was seen by me with a history of constipation and abdominal distention since birth. There was also poor appetite and failure to gain in weight. Though breast fed, the infant used to pass hard stools, once 3–4 days. Birth Wt – 7 lbs.

Physical examination revealed an undernourished infant weighing 11 lbs. The abdomen was distended. Huge coils of dilated colon were seen with visible peristalsis. On palpation, a few hard masses of faeces were felt along the colon.

Rectal examination: No soiling in the anal region. Sphinter was normal and the rectum was empty. Examination of the other systems were non-contributory.

The history of constipation from birth, the enlarged abdomen with the dilated colon, and the empty rectum made me suspect Hirschsprung disease.

To confirm this, a small barium enema was given and the x-rays of the colon taken in the A. P. and lateral views. These pictures are typical of Hirschsprung disease, showing the narrow segment below the dilated colon.

This condition is to be kept in mind since this is operable.

TWO CASES OF TRACHEO-OESOPHEGEAL FISTULA.

This is a condition where there is a fistulous communication between the trachea-oesophagus and is usually associated with oesophegeal atresia. The diagnosis is made by unusual amount of foaming at the mouth (mucus) at birth which persists after the initial aspiration of the mucus, and by inability to pass a soft catheter into the stomach. The fistula is usually of a small calibre. In

one case this was big enough to allow a catheter to pass through. Because a catheter can be passed into the stomach, tracheo-oesophageal fistula cannot be ruled out.

Case–1 : An infant of 12 months old was admitted with a diagnosis of tracheo-oesophageal fistula by the Obstetrician, because there was unusual amount of foaming at the mouth and he was unable to pass a catheter in the stomach.

On admission, there was considerable amount of foaming at the mouth. An attempt was made to pass a small soft catheter into the stomach. After several attempts, the catheter did pass into the stomach. The infant appeared to tolerate it well. When the baby was screened the catheter was visualised in the stomach. It was therefore considered that tracheo-oesophageal fistula could be ruled out.

However, the next morning the infant was still seen foaming at the mouth. So further x-ray studies were done using Lipiodol. Tracheo-oesophegeal fistula of typel was confirmed i.e. blind upper pouch with tracheo-oesophegeal fistula with the lower segment of the oesophagus with air in the stomach. At surgery, the diagnosis of tracheo-oesophegeal fistula was confirmed. This fistula was noted to be unusually large and this was closed. The catheter obviously passed down the treachea, through the fistulous tract into the oesophagus and finally into the stomach. It is important to bear this in mind, when faced with a similar problem and to use x-ray studies with lipiodol in all doubtful cases.

In some cases of tracheo-oesophegeal fistula, there may be vomiting, choking and cyanotic spells after feeding. There may or may not be " excessive foaming " at the mouth at birth. This is a rare type occurring in 1% of cases. At the same time, the condition is to be kept in mind for once this is recognised the operation is easy and the chances of post-operative stricture are rare.

The following case is an example.

Case–2 : An infant a few hours after birth was seen because of vomiting choking and cyanotic spells after feeds. To rule out a tracheo-oesophegeal fistula a catheter was passed into the oesophagus and it readily passed into the stomach and when flouroscoped catheter and air were visualised in the stomach. Since the symptoms were persisting, with catheter in the oesophagus, lipiodol was pushed into the stomach and an x-ray picture was taken. At that time there was simultaneous filling of the oesophagus and trachea. The dye was seen overflowing through the fistula into the trachea. At operation there was a small fistulous tract between the oesophagus and trachea. X-ray showed catheter in the oesophagus and simultaneous filling of the oesophagus.

A CASE OF DIAPHRAGMATIC HERNIA WITHOUT SYMPTOMS IN AN INFANT OF 9 MONTHS OLD

This case is of interest for most cases of diaphragmatic hernia in children where there is protrusion of the abdominal contents into the thoracic cavity through a defect in the diaphragm are recognised early in the neonatal period, because of symptoms of respiratory distress. In this case, the infant had bowel and spleen in the chest without symptoms.

Case : A 9 month old infant was admitted into the hospital because of abnormal findings in the chest during routine physical examination. During the previous two months before admission, he was seen five times in the out-patient department and was treated for two mild respiratory infections. At those times there were no symptoms referable diaphragmatic hernia. This infant was born after full term uneventful preg-

nancy and normal labour. Respirations were established spontaneously after birth. There was no cynosis or cyanotic spells. The neonatal period was uneventful. Development was normal.

Physical examination revealed a well developed, well nourished infant who did not appear ill. The apex beat was displaced to the right. There was diminished resonance on the left side. On auscultation, there was no wheezing, rhoncii or rales. Occasional gurgling noises were heard. Abdomen was soft. The liver was palpable below the right costal margin.

X-ray chest: Plain x-ray of the chest showed mediostinal shift to the right. Right lung was clear. In the lower 2/3 of the left chest ' bowel pattern ' was seen – collections of varied radiolucent areas.

A colon flurescopy: The colon filled readily and the splenic flexor the colon was seen to enter the chest.

On operation: There was a hernia through the foramen of Bochdelek. Spleen and the large bowel was in the chest. Viscus was returned and the defect was closed.

GALACTOCAEMIA.

This is rare inherited disorder of carbohydrate metabolism in which galactose which is derived from ingestion of lactose cannot be converted into glycogen in the liver. When neonatal jaundice is unexplained, one must think of galactocalmia and examine the urine for galactose.

When it manifests in infancy, there is loss of appetite, vomiting and failure to thrive. Enlargement of the liver, impaired mental development and cataract may be present.

Clinically:

1. Infants with galactocaemia develop normally for some time, although galactocaemia is present from birth if milk fed.

2. Feeding difficulties with occasional vomiting and diarrhoea appear within the first week or month.

4. Hepato-splenomegaly appear quickly. Subclinical early hepatic damage may be responsible for jaundice as continuation of physiological jaundice.

4. Nuclear cataract may develop at the same time as liver enlargment.

5. A significant degree of mental retardation is seen in most patients, in addition to failure of physical growth if the feeding with lactose is continued for an appreciable period of time.

6. Hypoglycaemic convulsions may occur even when total blood sugar is in the normal range.

7. X-ray Bone reveals osteoporesis.

8. Albumin and casts are frequent urinary findings.

Case:

A 4 month old infant was admitted because of failure to gain in weight. (Birth Wt. 5 lbs 8 oz. On admission 7 lbs – 10 oz.)

History of present illness: Full term. Normal delivery. Birth Wt. 5 lbs 8 oz. When started on milk formula, it did not agree with the infant. There was vomiting and dehydration. He was rehydrated with 5% glucose in water and sent home on the 8th day. The Wt. was 5 lbs 3 oz. There was no jaundice.

During the succeeding months there was a slow gain in weight. The appetite was good. Yet the gain in weight was slow. When the milk diet was increased he used to have spells of vomiting and diarrhoea.

At the age of 4 months, there was distension of the abdomen with moderate hepato-splenomegaly. There was four plus sugar in urine and fasting blood sugar - 165 mgm.

At 5 months, the reducing substance in urine was diagnosed as galactose and admitted into the hospital for further study. The child's weight was 7 lbs-2 oz. There was three plus sugar in urine. Fasting Blood Sugar was 129 mgm% and galactose 92 mgm%

X ray wrist revealed 2 carpel centres.

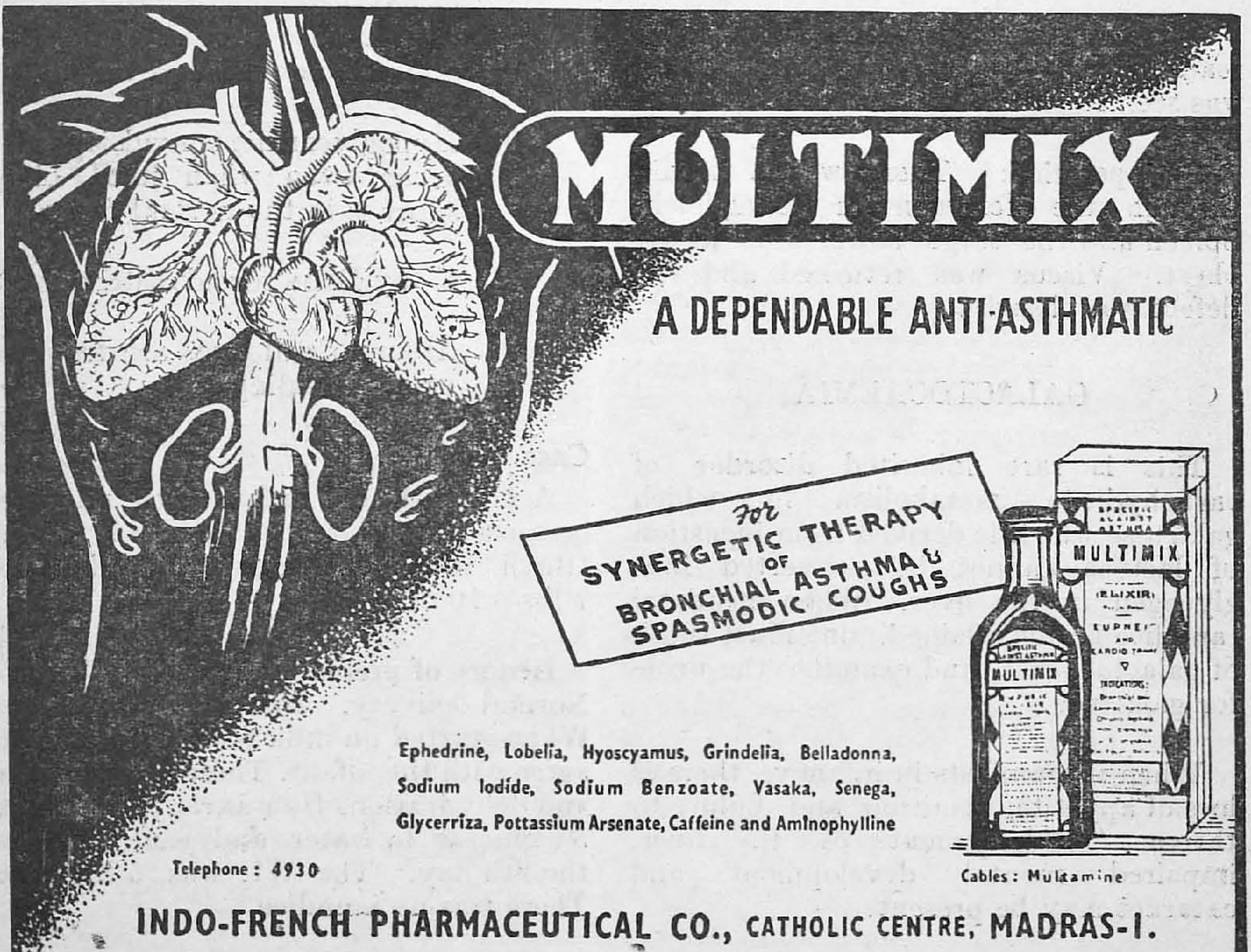
Child was started on nutramigen.

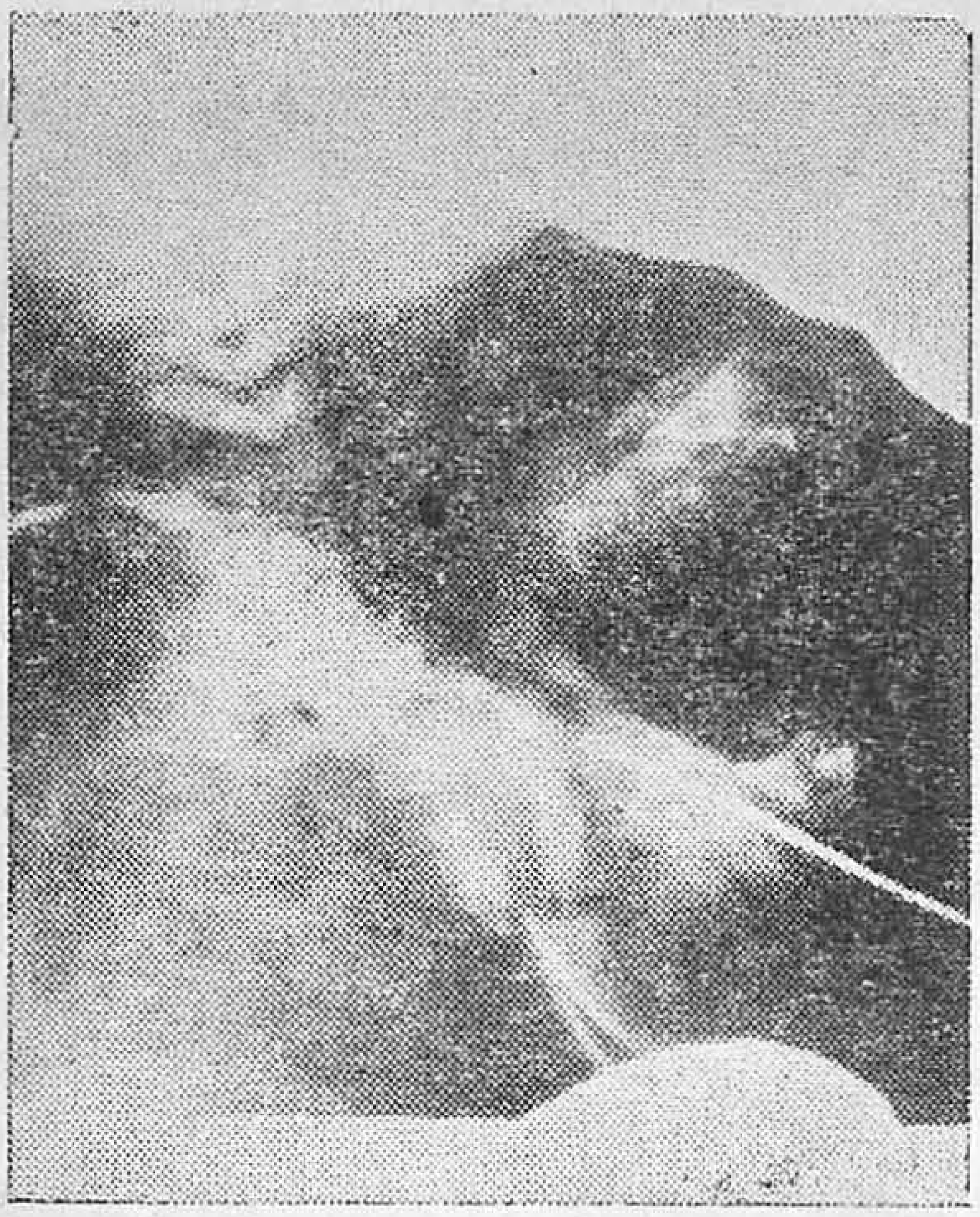
Gradually urine became normal as well as blood sugar.

Sent home on 40th day - Wt. was 8 lbs. 8 oz.

Nutritional status at the time of discharge was improved Liver was palpable, 2 1/2 fingers below the costal margin.

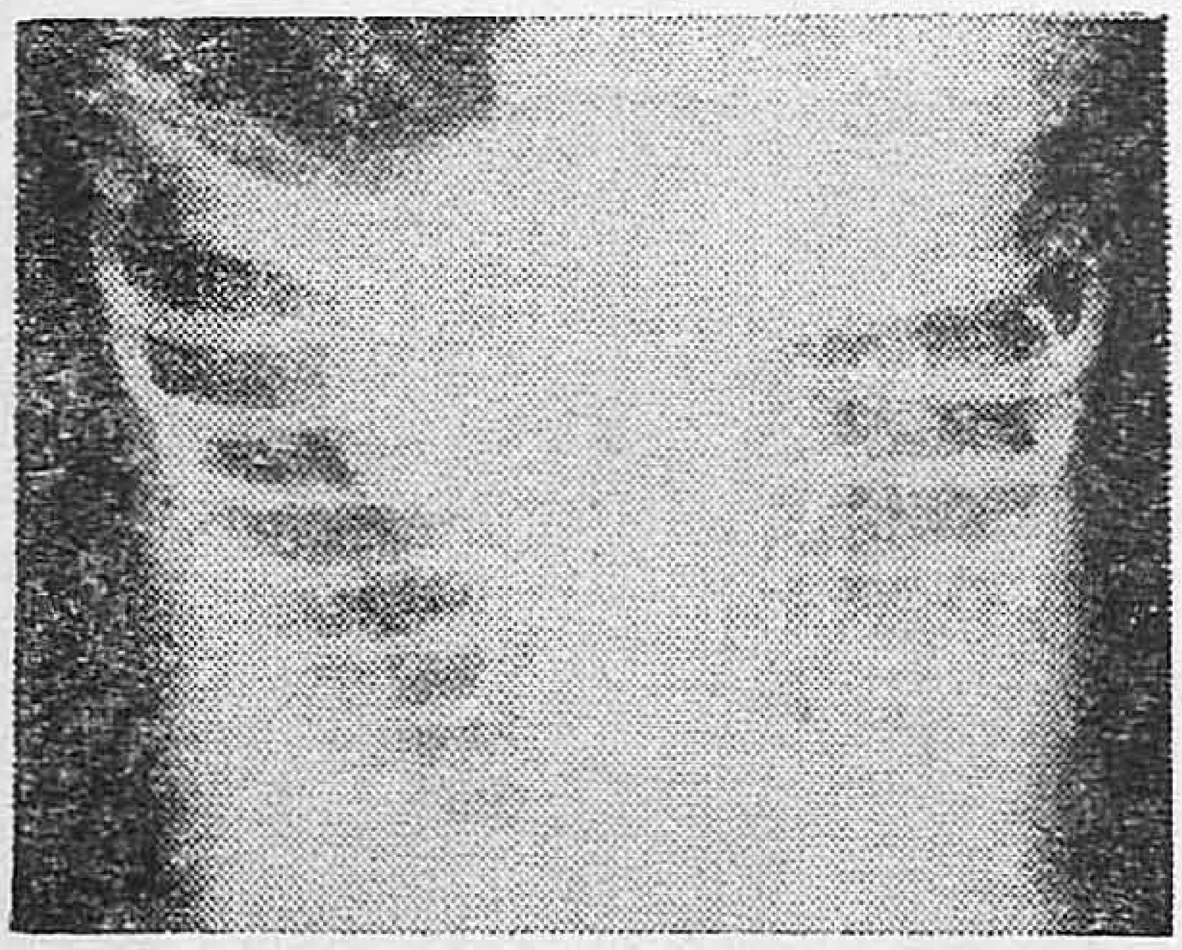
There were no neurological signs.





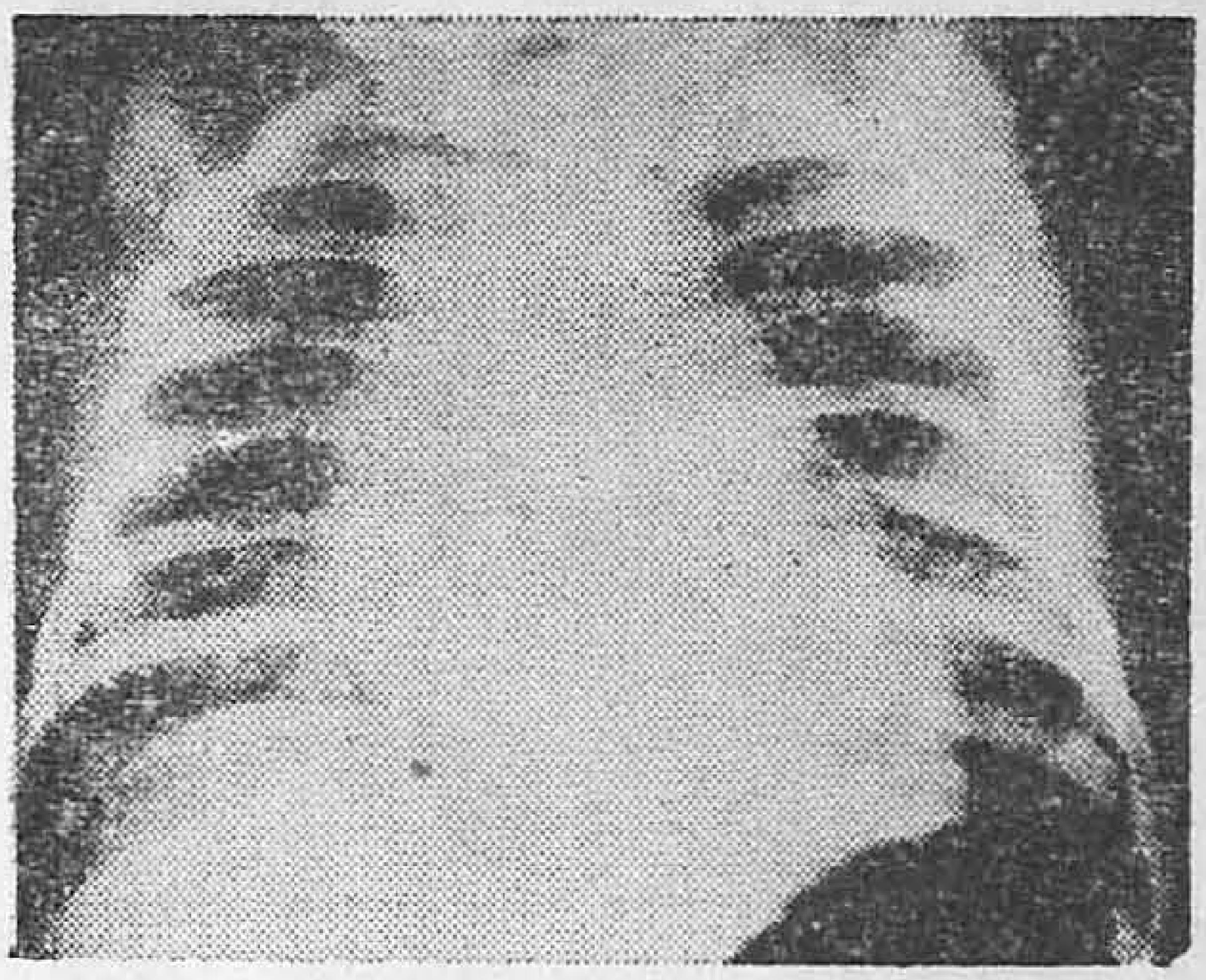
CHOANAL ATRESIA

X-Ray Showing probe in the nostril meeting the obstruction



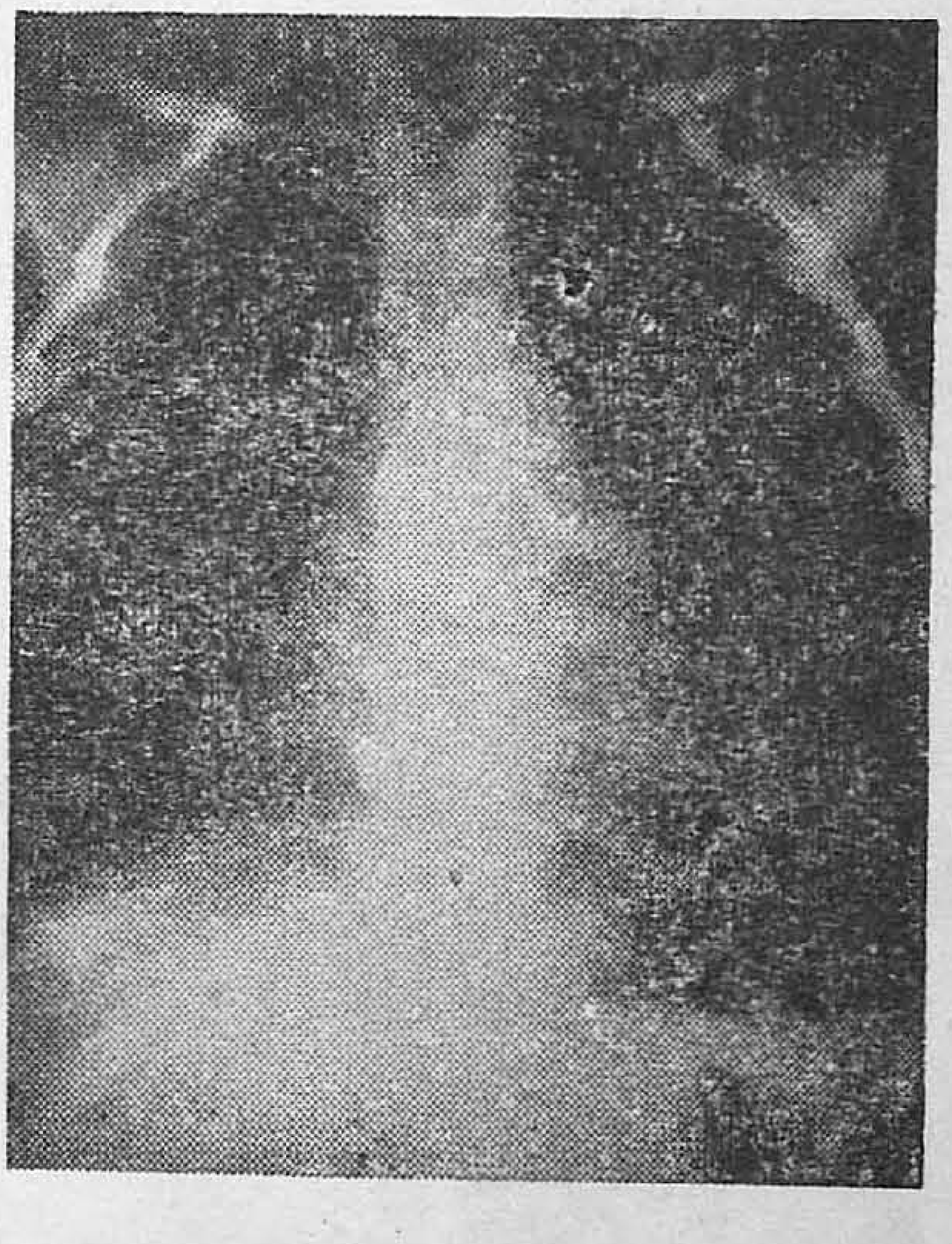
PULMONARY HYALINE MEMBRANE DISEASE

X-Ray of the chest taken 10 days later after the first X-Ray, showing partial clearing



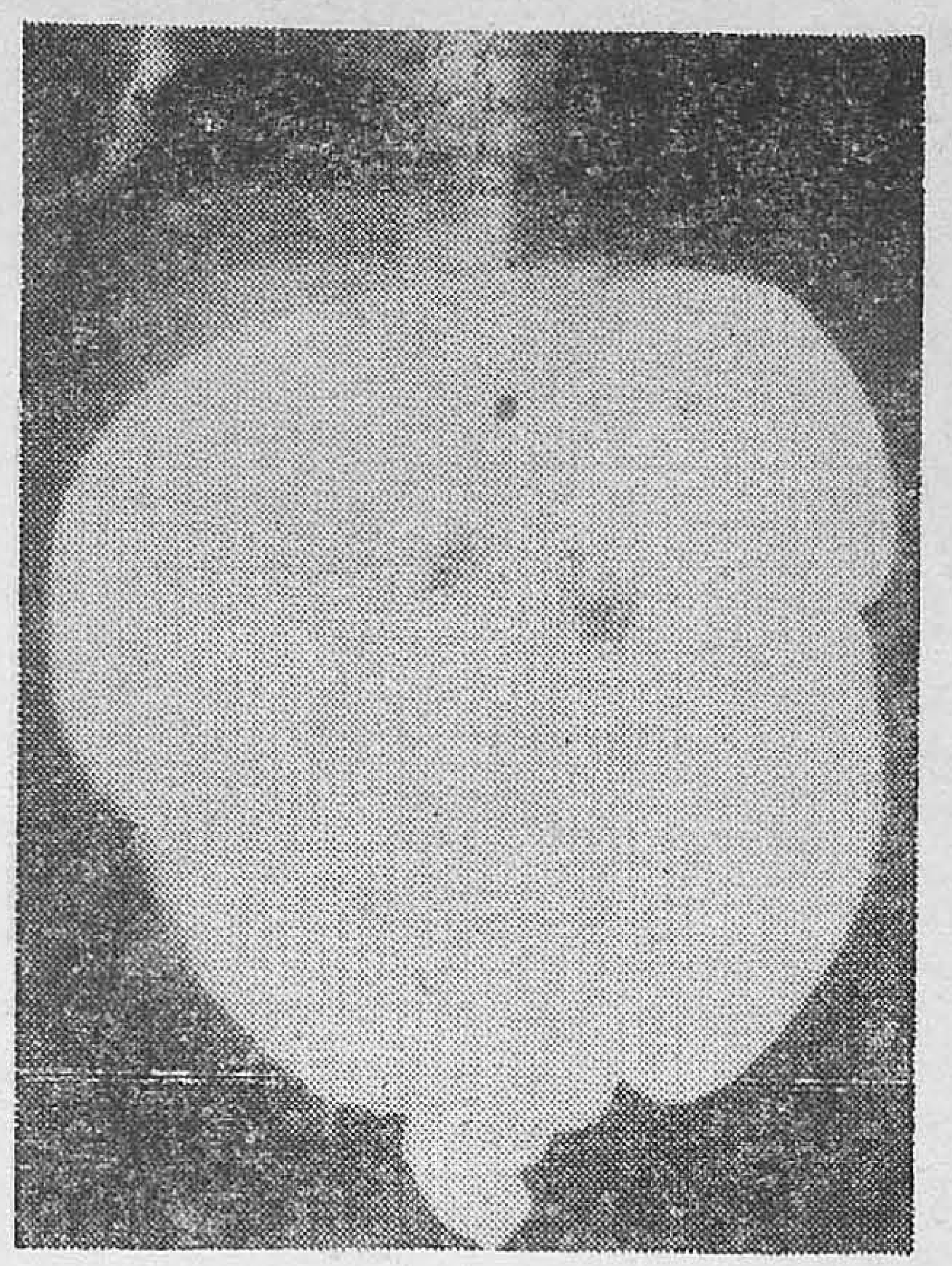
PULMONARY HYALINE MEMBRANE DISEASE

X-Ray of the chest showing generalised mottling throughout the lungs. (First X-Ray of the lungs).

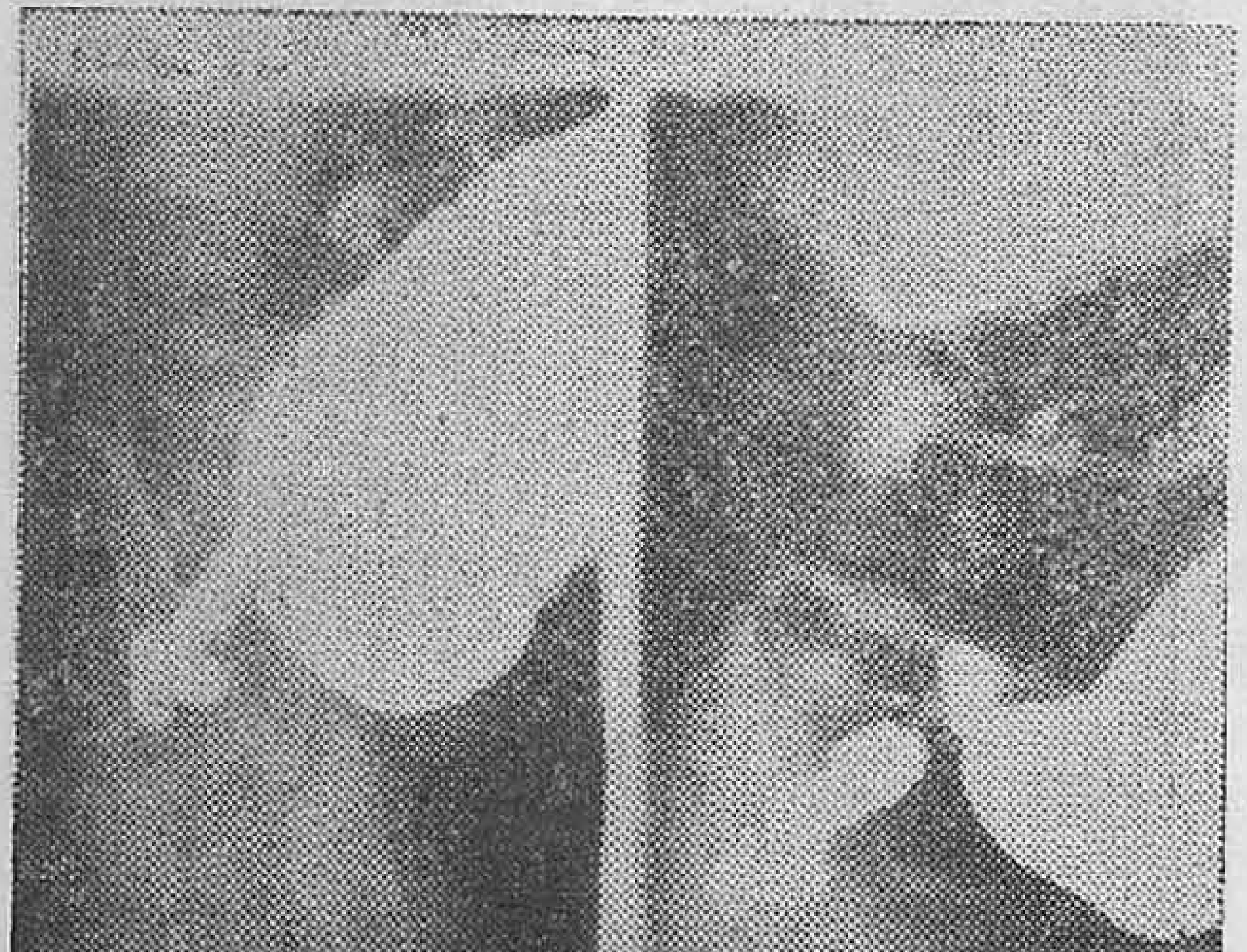


A case of foreign body in the bronchus stimulating bronchial asthma X-Ray of the chest - A. p. view showing a foreign body - a nail in the left main bronchus

A case of foreign body in the bronchus stimulating bronchial asthma.
X-Ray of the chest-Lat view showing a foreign body a nail in the left main bronchus.

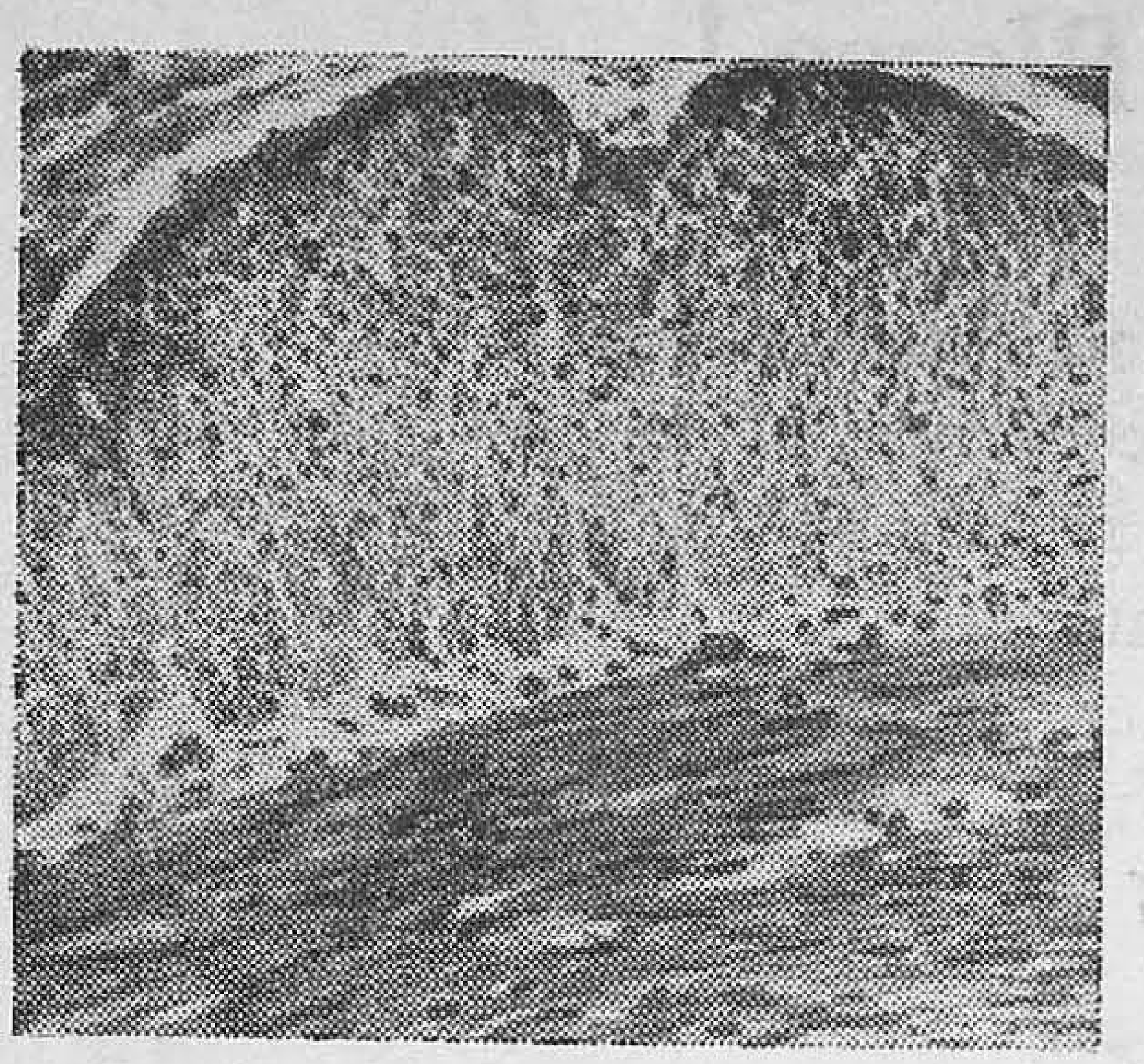


A case of Hirschsprung's Disease in an infant of 4 months.
X-Ray of the Colon A p. view after a small barium enema showing the narrow segment below the dilated colon.



A case of Hirschsprung's Disease in an infant of 4 months old.
X-Ray of the colon - oblique view after a small barium enema showing the narrow segment below the hugely dilated colon.

NORMAL COLON
Section of the colon showing the ganglion cells between the inner circular and the outer longitudinal muscle fibres in a normal colon (dilated colon)

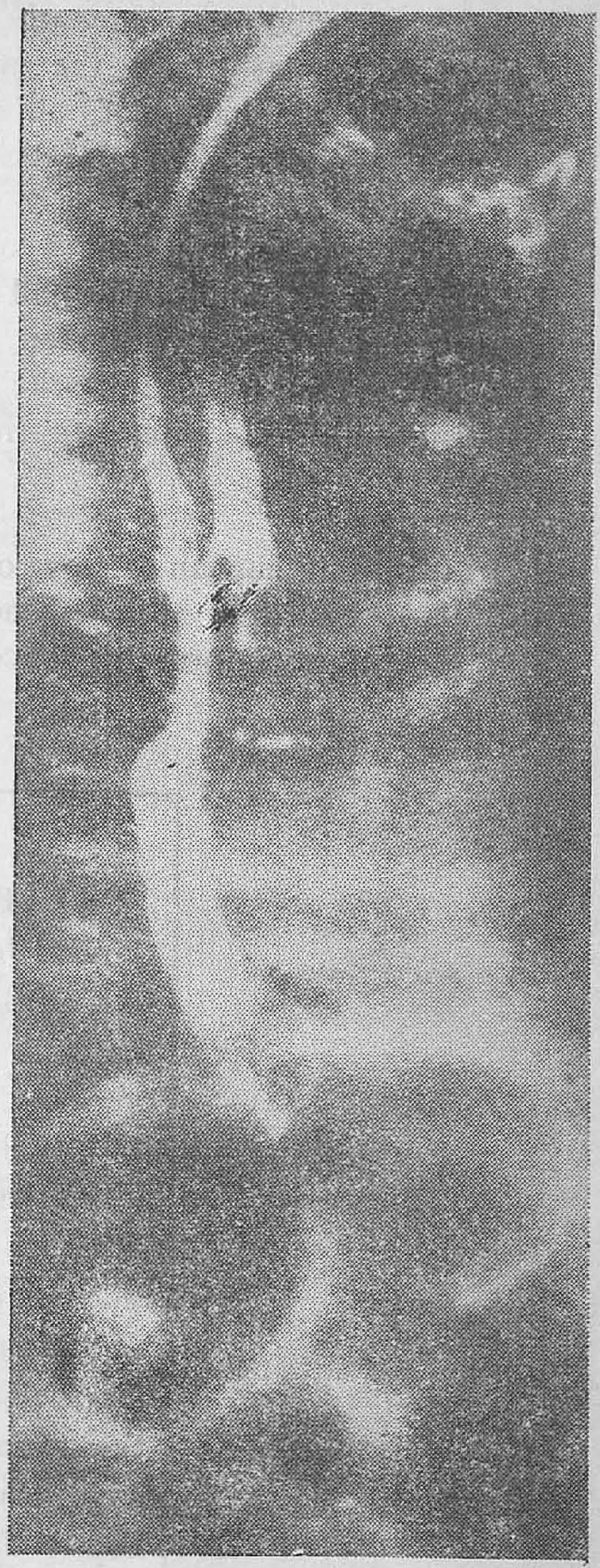


A CASE OF HIRSCHSPRUNG DISEASE

Section of the narrow segment of the colon showing the absence of ganglion cells between the inner circular and outer longitudinal muscle fibres in a case of Hirschsprung disease.

A RARE TYPE OF TRACHEO-OESOPHEGEAL FISTULA.

X-ray of the chest taken with a catheter in the oesophagus and lipiodol pushed into the stomach. There is simultaneous filling of the oesophagus and trachea with a small fistulous tract between the oesophagus and trachea.



A CASE OF DIAPHRAGMATIC HERNIA IN AN INFANT OF 9 MONTHS OLD

X-ray of the chest after a barium enema-flexor. Splenic of the colon is seen in the left thorax. There is mediastinal shift to the right.

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Managing Editor.

NEED FOR CAUTIOUS APPROACH IN THE USE OF THE PENICILLIN

The Press Release issued by the World Health Organisation is reproduced, as per the instructions of the Director of Medical Services, Madras, for the information and guidance of the members of the Indian Medical Association and other members of the medical profession, in order to ensure cautious use of Penicillin by them.

WORLD HEALTH ORGANISATION
Regional Office for South-East Asia
New Delhi, 26th Dec. 1958.

Press Release SFAR 576 :

Adverse Reactions to Penicillin

Warnings against indiscriminate use of penicillin have been issued by experts of the World Health Organisation following a comprehensive study of reports on adverse reactions to the drug.* The number of these reports has been steadily increasing in step with the fabulous expansion in pencillin production from a modest 29 pounds in 1943 when the drug first came into use, world production has now grown to 750 tons annually.

This growth, unprecedented in the history of pharmacology is further illustrated by the fact that today pencillin, together with the 400 other, less common antibiotics so far developed, represents half the value of the world's total pharmaceutical production.

In relation to this the number of toxic and allergic reaction has so far been exceedingly small. It is nevertheless estimated that by 1957 about 1000 fatal reactions to penicillin treatment had occurred in the United States of America alone and million injections can be expected.

Reactions may however become more frequent in the future as an increasing number of people are using more and more penicillin and gradually are being sensitized to this antibiotic. At present time, however the risk involved in using penicillin is not greater than that involved in the use of any other drugs and the tremendous good which pencillin is doing in curing diseases and preventing death should not be overlooked. Further more simple precautions may prevent some of the serious consequences unless prescribed by a physician. In this connection the WHO experts point out that severe penicillin reactions occur after repeated

* Review by O. Idosc, R. R. Willcox (WHO consultants) and T. Guthe (Chief, Venereal Diseases and Treponsematoses Section, WHO Geneva) Published in the WHO Bulletin Vol. 19, No. 3, 1958.

exposure to the drug, and that many cases of severe reactions can be attributed to previous unnecessary use of antibiotics.

Secondly penicillin should not be used against minor infections such as the common cold, for which it is ineffective or not more effective than other drugs.

Thirdly, self medication with antibiotics is dangerous and be avoided. The addition of penicillin to tooth paste, chewing gum etc., is 'indefensible' the experts say. Penicillin ointments should also be avoided.

According to the study, patients with a personal or family history of allergy are most likely to develop severe adverse penicillin reactions. In general, reactions are most common in adults between 20 and 49 years of age; they are rare in children under 12, and the frequency decreases rapidly with increasing after 50.

Another reason for restricting the use of penicillin is the development of resistance to antibiotics in staphylococci. These are among the most common micro-organisms and are found in the noses and throats of many healthy persons. They can cause boils, sore throats and even pneumonia in weak and sometimes in healthy people. Most strains of staphylococci were susceptible to penicillin when the drug first came into use, but hardy and more resistant strains emerge and have become a menace in hospitals where they sometimes cause delay epidemics among children and infants.

In conclusion, the WHO study emphasizes the fact that Penicillin still holds the field as 'the queen of drugs' despite the adverse effects it may sometimes produce. However 'not unlike many queens of the past, she may retaliate on those who do not respect her powers.'

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I. M. A. Madras State Branch.